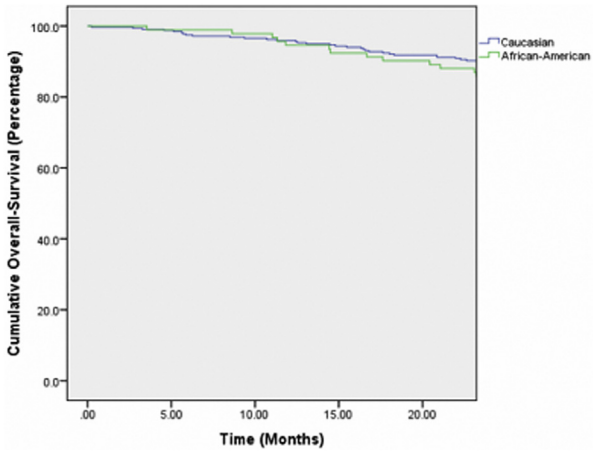

Figure 2. Overall survival among Caucasian patients compared to African American patients during the 24-month follow-up period (Log Rank=0.25).

January 2009 and May 2013. Comparisons of continuous variables between the two groups were assessed using student t-test and Wilcox log rank sum test; Chi-square test was used for categorical variables. Kaplan Meier curves were performed to assess survival and disease progression over time.

Results: During this time 92 AA and 317 C patients underwent ASCT. Median follow-up was 721 days in AA (IQR, 392–1103) versus (vs.) 735 days in C (IQR, 375–1124). The mean age of AA at time of transplant was significantly lower than that of Caucasian pts (55.67 ± 8 years vs. 57.74 ± 8 years, p = .032). Durie-Salmon stage distribution was similar in two groups with about 60% of patients in stage III at diagnosis. Cytogenetic risk distribution was similar in two groups: (AA vs. C) standard risk 82% vs. 86%; intermediate risk 16% vs. 10%, p = 0.17; high risk 1.5% vs. 4%, p = 0.34. Disease status at the time of transplant was CR in 8% AA vs. 21% C (p=0.03), VGPR in 42% AA vs. 29% C (p=0.66) and PR in 34% AA vs. 33% C. The median time from diagnosis to ASCT in AA was significantly longer than that for C {288 days; IQR (216.75–677.50) vs. 213 days; IQR (173.50–371.50), p= <0.001}. Induction therapy prior to transplant consisted of novel agent (proteasome inhibitor and/or immunomodulatory agent) based regimens in all patients. Median number of cycles of induction therapy was 5 (range 4-16) vs. 4 (range 3-22) cycles in AA vs. C. Preparative regimen for ASCT was Melphalan 140mg/m2 in 31% vs. 26% (AA vs. C) and Melphalan 200mg/m2 in the rest. Rate of disease progression after two-year median follow-up was higher in the AA pts 39% vs. 27%, OR 1.75 (1.08-2.85), p = 0.02 (Figure 1). There was no difference in the overall survival in two groups after median follow-up of two years 86% vs. 90%, OR 1.47 (0.73–2.93) p = 0.27 (Figure 2).

Conclusion: AA pts are diagnosed with MM at a younger age as compared with C population. Time from diagnosis of MM to referral to ASCT in the AA patients is longer than in the C. In addition, the rate of disease progression post-ASCT is higher in AA pts.

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CXCR4 Expression in Mantle Cell Lymphoma and Mobilization with Plerixafor for ASCT Does Not Negatively Impact Progression-Free Survival

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Introduction: Mantle cell lymphoma (MCL) is a rare B-cell malignancy comprising 5-10% of all non-Hodgkin lymphoma cases. Autologous hematopoietic stem cell transplant has emerged as a viable therapy for many patients with MCL. An important step in the harvesting of peripheral blood hematopoietic stem cells (HSC) is mobilizing $CD34^+$ HSC to the peripheral blood. The novel compound plerixafor is an antagonist of CXCR4, and has been successful in mobilization of HSC for autologous stem cell transplants for non-Hodgkin lymphoma. Increased mobilization of HSC in peripheral blood carries the risk of increased mobilization of lymphoma cells. Since MCL expresses CXCR4, we sought to determine whether administration of plerixafor can adversely affect outcome in transplantation. We report survival results of autologous peripheral blood stem cell transplants (ASCT) for patients with MCL who were treated with and without plerixafor for HSC mobilization prior to ASCT at Mayo Clinic.

Methods: The present study is a retrospective cohort study of all adult patients who underwent ASCT for treatment of MCL at Mayo Clinic from February 1993 to December 2013. Patients were divided into two cohorts: (1) patients treated with plerixafor for HSC mobilization prior to ASCT, and (2) patients not treated with plerixafor prior to ASCT. The primary outcome was relapse of MCL. Overall survival from diagnosis and overall survival from transplant were also analyzed.

Patients: From 1993 to 2013, 169 consecutive patients underwent ASCT; 55 patients received plerixafor for HSC mobilization prior to ASCT, and 114 patients did not receive plerixafor. The median ages at MCL diagnosis and ASCT for the plerixafor cohort were 57.7 years and 58.4 years, respectively, compared with 56.6 years and 58.1 years for the non-plerixafor cohort.

Results: The average $CD34^+$ HSC harvest with plerixafor was 5.4×10^6/kg compared to 4.9×10^6/kg without plerixafor ($p = 0.13$). Median progression-free survival from ASCT was 3.4 years in the plerixafor cohort compared with 3.6 years in the non-plerixafor cohort ($p = 0.69$). Median overall survival from ASCT was not reached in the plerixafor cohort compared with 5.7 years in the non-plerixafor cohort ($p = 0.95$). Median overall survival from MCL diagnosis was 8.9 years in the plerixafor cohort compared with 7.6 years in the non-plerixafor cohort ($p = 0.71$).

Conclusions: ASCT for treatment of MCL performed at Mayo Clinic resulted in no statistically significant differences in progression-free and overall survival between patients receiving and patients not receiving plerixafor for $CD34^+$HSC mobilization prior to ASCT. The results of this study indicate that HSC mobilization using plerixafor is not associated with decreased progression-free or overall survival from ASCT in MCL. There appears to be no clinically significant mobilization of lymphoma cells associated with plerixafor mobilization of HSC.

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Engraftment Failure or Aplastic Anemia: A Case Report

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Secondary engraftment failure is defined as neutrophils increase to ≥ 0.5 x 10 ⁹/L and subsequently decrease to a lower level until additional treatment to obtain